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ORIGINAL ARTICLES.

A SIMPLE LACRIMAL SYRINGE.

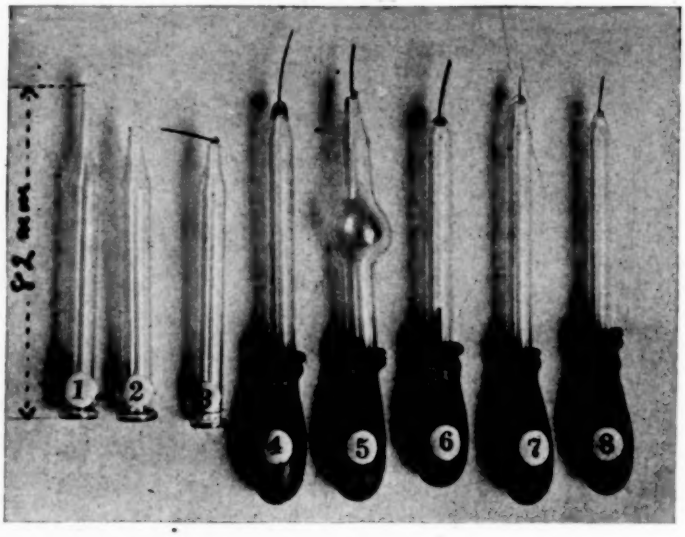
By W. E. Shahan, A.B., M.D.,

ST. LOUIS, MO.

This syringe has shown a high degree of efficiency in practical application, and is so simply constructed that it may be improvised by anyone from materials in common use. It consists essentially of an ordinary glass dropping tube into the small end of which is fused a piece of platinum tubing. Platinum or platinum-iridium tubing has become generally available in the form of hollow needles for hypodermic use, use in intra-muscular injections, etc. These needles are carried in stock by druggists and instrument dealers and can be obtained in various sizes. By means of a fine file it is easy to cut them into lengths suitable for use as lacrimal syringe points. The end of such a point that is to be fused into the glass should be filed to a smooth cross section by drawing the file lightly over it, and the opening freed from overhanging edges by twirling the point of a pen knife in it. The tip should be filed or whetted down to a blunt round termination (as seen under a magnifying lens) so as to remove all jagged edges that might lacerate the walls of the canaliculus or nasal duct.

After the point has been prepared the next step is fusing it into the glass tube. Glass dropping tubes are easily obtainable in various sizes. By making a scratch with a file on the small end of such a tube as is shown in Fig. 1, the tip can be broken

off, as is shown in Fig. 2, so as to give a somewhat firmer base for the point to be fused into. This is then held tip downward in a Bunsen or large alcohol flame until the opening closes to a size a little larger than the platinum point to be used. The tip is then held upward and the platinum point balanced in it at any angle as shown in Fig. 3. The point so balanced is then carried over into the flame. It almost immediately becomes white hot and sticks to the glass. As soon as it sticks it can be held point downward in the flame, and, as the glass softens, gradually sinks of its own weight to a more or less vertical position, and becomes firmly fused into the glass. The position of the point can be varied at will by turning the glass tube one way or another be-



tween thumb and finger. After the union of the glass and metal is complete the syringe should be elevated gradually from the flame, and hastily tucked into a wad of cotton, so as to anneal the glass by causing it to cool slowly. The whole operation requires only a few minutes. The glass is not simply banked around the metal but is tightly coherent to it so that if the point is violently broken away from the glass, it does not come away clean, but has particles of glass still sticking to it. This is probably explained by the nearly identical coefficients of expansion for glass and for platinum. The coefficient of expansion of glass is 0.0000086 and that of platinum is 0.0000088 per unit of length per degree centigrade (Ganot). This explains why the glass

does not crack around the metal during marked changes in temperature, as in boiling. Gold points have been used with a fair degree of success in the same way as the platinum ones. But owing to the higher coefficient of expansion of gold (0.0000146 per linear unit per degree centigrade), troublesome cracking of the glass was often experienced. Gold also melts at a temperature much lower than of platinum, and sometimes liquifies during the attempt to fuse it into the glass. For both these reasons silver is still less desirable.

This furnishes a series of neat simple syringes such as are shown in Figs. 4, 5, 6, 7, and 8. The smallest size (8) is of special value in injecting solutions into undilated or very small canaliculi for purposes of diagnosis or anesthesia preliminary to probing or operative procedures. The small sharp points with broad glass shoulder (Fig. 7) can be used when it is desired to block the punctum with the glass to prevent regurgitation.

If it is desired to increase the capacity of the tube, a bulb can be blown in it (Fig. 5). Larger points (Fig. 6) are used in large canaliculi or in canaliculi that have been slit.

These syringes are easily sterilizable by boiling in carbolic acid or other solution, or by keeping in almost any antiseptic solution. If the glass should be broken it is an easy matter to fuse the point into a new glass tube. If the point becomes clogged, as the smaller ones are apt to, the obstruction can be burned out by sticking the point into a flame and allowing it to become white hot. This is one of the great advantages of platinum points over points of gold and silver, as gold and silver melt at a temperature only a little higher than the necessary softening point of glass. The rubber caps ordinarily used on dropping tubes are of sufficient capacity for most purposes. If larger ones are desired they can be obtained at photographic stores in the form of shutter bulbs. Such a pressure arrangement as this can be handled with greater facility and more perfect control than the piston syringes in general use.

It is perhaps unnecessary to add that this form of syringe can be used in irrigating the anterior chamber or in injecting fluids into other cavities, as the accessory nasal cavities, or, with very sharp tips, in making subconjunctival or even hypodermic injections.

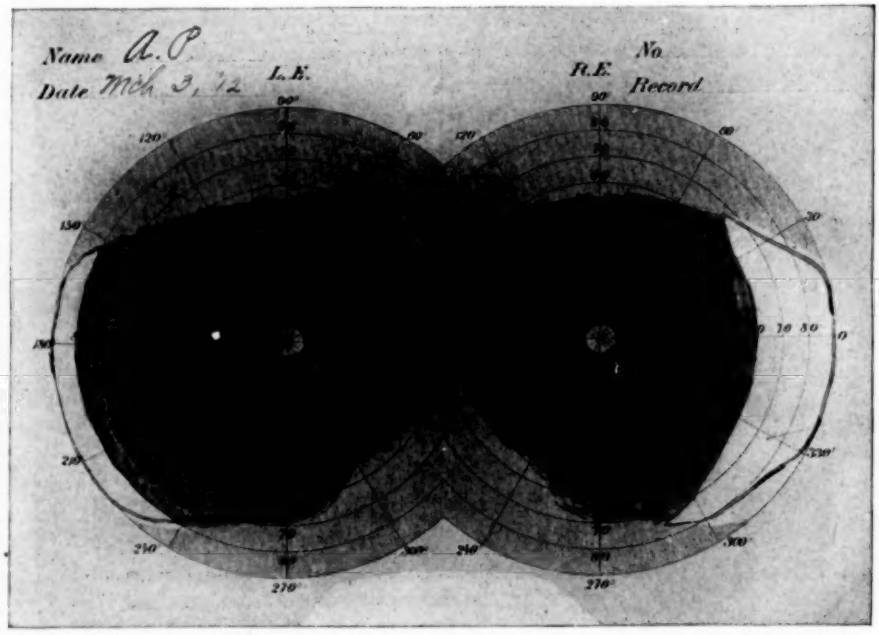
RETINITIS PIGMENTOSA SINE PIGMENTO.*

REPORT OF A CASE.

By J. F. Shoemaker, M.D.,
ST. LOUIS, MO.

This case is one of degeneration of the retina, with marked concentric contraction of the visual fields, which I believe belongs to that class of cases which is designated as *retinitis pigmentosa sine pigmento.* At any rate it seems worth reporting as there are a number of interesting points connected with it.

A. P., aged 26, consulted me first on June 25, 1910, complain-



ing that during the past three years or more he could not see well, especially at night. He had suffered a great deal with headache previously but was free of it at that time. His vision was found to be 18/38— in the right eye and 18/48 in the left and he could read Jaeger No. 1 at 6 inches with either eye. His vision could not be improved with glasses. Ophthalmoscopic

*Read before the Ophthalmic Section of the St. Louis Medical Society, February 7, 1912.

examination showed posterior capsular cataracts in both eyes. This seemed sufficient to account for his poor vision, and he was given a collyrium for local use; 1/12 grain bichloride of mercury, internally, three times daily, was ordered and he was asked to return in one week for further examination and observation. This he failed to do and I did not see him again until November 18th, 1911, when he returned and complained that during the two weeks previous his vision was much worse. His vision at this time was 13/40— with the right eye and 13/70 with the left unimproved with glasses, and he could only read Jaeger No. 7 with the right eye and Jaeger No. 12 with the left. His visual fields were greatly contracted, as evidenced by his peculiar manner of moving about and the fact that he bumped into large objects quite frequently. His fields were charted at a later visit and were found to be concentrically contracted to within five degrees of the fixation point for form while the fields for blue and red were practically the same. In addition to the central vision of five degrees from the point of fixation he can also see large objects in the temporal fields corresponding to about from the 60th to the 90th degrees in the right eye and 80th to 90th degrees in left. Examination with the ophthalmoscope at this time showed, in the right eye, posterior capsular cataract, which is about the same as it was twenty months ago; clear vitreus; optic papilla slightly pale, especially on the temporal side; and the retinal blood vessels markedly contracted. There are no deposits of pigment in the retina to be seen and no atrophic spots of choroiditis. The left eye shows practically the same condition except that there is in addition a small opaque mass in the vitreus. Inquiry into the family and personal history elicited the following information: Father, mother and two sisters living, none of whom have ever had any defects in their vision. He does not know of any of his relatives having had any visual disturbance similar to this. No blood relationship between his parents. The patient states that when he was five or six years of age he had some kind of an illness (does not know what), after which he could not see well at night. This lasted for some time but finally disappeared and his vision at night seemed all right. He says that as an older boy he rode a bicycle and frequently rode at night and could see as well as other boys. Both his distant and near vision were good until he was about twenty years of age when he noticed that he could not see so well at night or in a poor light. Later his vision was

not so good at day time as it had been previously. States that he frequently noticed flashes of light and momentary obscurations of vision at that time. He had an attack of gonorrhœa about this time but aside from this he has had no illness of any consequence since he was five years of age. Denies luetic infection. At present he complains of frequent attacks of what he describes as feeling as if he were not right in his head, or as if he were drunk, although he drinks but very seldom and then only a glass of beer. The treatment the past two months has been the administration of strychnin and K. I. He thinks his vision is some better and continues his work in a shoe manufactory. His vision at present with a bright light is 18/48+ with the right eye and 18/60— with the left, and he reads Jaeger No. 2 with the right eye and Jaeger No. 12 with the left.

The night blindness, concentric contraction of the visual fields with comparatively good central vision which, were it not for the cataracts, would doubtless be close to normal, the arcs of vision in the periphery of the temporal fields, pointing as they do toward ring scotomata earlier in the history of the case, the appearance of the optic nerve heads, all taken together speak in favor of our considering this as a case of retinitis pigmentosa without the pigment. If it be such, there are these two unusual features about it. In the first place most authors agree that reinitis pigmentosa is in the majority of cases an hereditary disease and in many cases it is attributed to consanguinity on the part of the parents. There was no blood relationship in the case of this patient's parents and so far as I am able to learn, no other case of ocular disturbance of this nature in either the immediate family or in any of their kin. Secondly, the rapid progress of the disease until the visual fields are but little more than the points of fixation is quite unusual. As a rule the disease is a slowly progressive one, reaching this stage only after the fortieth or fiftieth year.

The ætiology of retinitis pigmentosa is not definitely known although heredity seems to play a very important part in a large percentage of the cases, and consanguineous marriages appear responsible for many others. Snell believes that if all cases were investigated thoroughly very few would be found in which at least one of these factors was not present. He thinks, however, that from what data we have we are not justified in believing that consanguinity can produce the disease, except by increasing an hereditary tendency. E. V. Knape is of the opinion

that retinitis pigmentosa has as its predisposing cause an obliterating endarteriitis of the choroidal vessels, due to congenital tendency, and that some disease of the eye, a specific fever, or syphilis may act as the exciting cause. In harmony with this theory Chaillous reports a case in which this ocular defect together with defects of speech and hearing occurred distinctly after an attack of diphtheria, which had also been followed by paralyses.

There are several theories as to the anatomical changes responsible for the blindness incident to this disease. The first, held by Nettleship, Gonin, Knape, and others, is that the disease begins in the choroid and thus affects the nutrition of the retina, producing the atrophy or degeneration. They point out that the venæ vorticosæ pass out of the globe at the equator and it is here where the degeneration of the retina begins in this disease, spreading toward the macula and also toward the periphery. As already stated, Knape believes that the choroidal trouble is of the nature of an endarteriitis obliterans. The second theory, championed by Lister, Hancock, Ginsberg, and others, is that the degeneration begins primarily in the nervous tissues, rather than in the vascular system; the concentric effect corresponding with the nerve distribution, rather than the vascular. R. W. Doyne, who has recently called attention to a zone of greyish infiltration inside the pigmented zone, in this disease, and also described opalescent threads in the vitreous just in front of and covering nearly all of the retina, "much like the gossamer seen on an autumn morning over a turnip field," advances still another theory. He does not believe the disease begins in the choroid, nor that it is a simple atrophy of the retina, but thinks that it is due to an exudation which chokes the vessels in the disc, diminishing their size and causing the peculiar color of the disc; also perhaps the *drusen bodies* and the greyish color of the retina referred to. Probably this exudate is partially carried off through the lymph channels accompanying the venæ vorticosæ and thus chokes the channels, causing the pigmentation to begin at this site, the equator of the eye ball. Where the exudate comes from, he states he does not know.

There is no record of any miscroscopic examination of retinitis pigmentosa sine pigmento, but a few cases of true retinitis pigmentosa have been studied anatomically and the findings are of considerable interest as throwing light upon the pathogenesis. They rather support the theory that the disease begins in the

choroid since, as a rule, there are marked sclerotic changes in the choroid with more or less atrophy of the choriocapillaris. In advanced cases the nerve tissue of the retina is entirely destroyed and replaced by fibrous tissue. In less advanced cases the degeneration is most marked in the outer layers of the retina which are dependent upon the choroid for their nutrition. In the parts of the retina where the pigment has been deposited the rods and cones and most of the other nerve elements are always destroyed; moreover, the degenerative process is much advanced in the non-pigmented zone adjoining the pigmented part. This shows that the pigmentation does not take place until after the complete destruction of the retina. In no case have any evidences of inflammation been found, proving that the process is a degenerative one and not an atrophy following inflammation.

No treatment has been found which is effective in stopping the progress of this disease. Improvement has been noted after the administration of such remedies as strychnin, but it has always proved to be but temporary. The vision has seemed to improve materially in a number of cases while the patients were being fed fresh retinas of animals, but always relapsed as soon as the feeding was stopped. The tendency is toward complete blindness, although many cases retain some central vision as long as they live.

EXOPHTHALMOS FROM A BONY TUMOR GROWING FROM THE NASAL WALL OF THE LEFT ORBIT.*

By M. H. Post, M.D.,

ST. LOUIS, MO.

Miss —, 19 years old, consulted me October 3rd, 1911, having been sent to me by Dr. C. M. Sneed, of Jefferson City, Mo.

She stated that her eyes began getting "weak" about two years ago. A year later she began to see double, one object being almost directly above the other. Alarmed at this condition she sought the help of "Dr"—, "Optometrist", who gave her glasses.

Some time last March, about nine months ago, she noticed

*Read at meeting of St. Louis Ophthalmic Society, Dec. 18, 1911.

that the upper lid of the left eye drooped a little. On account of this, and because the glasses had not helped her, in June she consulted Dr. Sneed, who changed her glasses, and gave her medicine internally.

Up to this time, nearly a year and a half after the first symptoms, no intelligent effort had been made to diagnosticate the underlying cause.

The third of last October, at the suggestion of Dr. Sneed she consulted me. When I saw her she complained of pain in the left side of her head upon exertion. She was wearing glasses given by Dr. Sneed which were O.D.+.75 D.s.⊃+0.12 D. cyl. axis 150°; O.S.+1. D. cyl, axis 165°. With them O.D. V. 20/12 O.S. V. 20/⅓9 (?).

The ophthalmoscope showed the disc O.S. possible a little hyperæmic as compared with that of O.D. but not markedly so. The globe was thrust somewhat forward, outward and a little downward. By a voluntary effort she could elevate the upper lid of the left eye almost normally.

Hyperduction and adduction of the globe were nearly lost, The abducens seemed to act normally. The pupil was moderately dilated. The condition was that which is produced by a paralysis or a paresis of the third nerve. Unfortunately I made no test of the accommodation.

Upon palpation, while the tension of the globe was not raised, the globe did not recede under pressure as it normally does, but offered resistance as if it were pressing against a hard substance. No tumor could be felt by the fingers.

There was no pain upon pressure, and no inflammatory swelling of the tissues of the orbit, as there probably would have been if the conditions had been caused by a sinus trouble.

I diagnosticated the condition as one in which a tumor to the nasal side of the globe and well back in the orbit was pushing the eye out of place and was pressing on the third nerve.

In the hope that the tumor might be luetic the patient was referred to Dr. Tiedemann for a Wassermann. Unfortunately his report was negative. In the hope, that notwithstanding the lack of inflammatory symptoms, it might be due to some form of sinusitis she was next sent to Dr. Sluder whose findings were negative. Dr. Sluder and I decided that an X-ray picture should be taken, and we referred her to Dr. Carman. He reported a bone tumor well back in the orbit.

Still being at a loss for a positive diagnosis as to the character of the tumor, we thought mercury and iodide of potassium

should be administered for a time, in the hope that notwithstanding the probability that it was not luetic it might subside under this treatment, as some cases have been reported to do. On this account I sent her home on October 16th, at the same time writing to Dr. Sneed suggesting such a course. He followed this suggestion, though he wrote me that before she was sent to me this had been done.

November 24th, about six weeks later, the patient returned to my office very much discouraged and disheartened. I had suggested to Dr. Sneed in my letter when she returned to Jefferson City, that if our treatment was not soon followed by improvement, we had best have Dr. Mudd examine the case, knowing that his wide experience in tumors would be helpful and wishing in case an operation were necessary, that he should perform it. Dr. Mudd examined the case and considered that an operation was necessary, but thought it would be well to crowd the iodide of potassium under our own observation. Accordingly on November 27th, 1911, I prescribed ten minims of a one-half solution of iodide of potassium in water, increasing this rapidly until on December 4th she was taking seventeen and a half grains three times a day. At this time she complained of soreness of the nose and throat and I stopped the treatment.

On December 6th she reported, saying that she was no better and that Dr. Sneed had given her the same medicine. We thought an immediate operation best, and with her mother's consent, and after consultation with Dr. Sneed over the telephone, it was decided to remove the tumor at the earliest opportunity. This was done December 8th. Dr. Mudd removed the eye, the contents of the obrit, the periosteum of the orbit and after that the tumor. The tumor was located about ½ inch back in the orbit, and extended almost to the sphenoidal fissure. It seemed to originate in the ethmoid cells and it carried before it part of the lacrimal bone and the lamina papynaceæ of the ethmoid.

The question of removing the tumor without sacrificing the eye was considered, but as there was a possibility that the tumor was malignant and we had reason to believe that it extended very far back into the orbit, which proved to be the case, it seemed best to perform the operation which was selected. The patient is now doing well.

MICROSCOPICAL EXAMINATION OF DR. POST'S TUMOR REMOVED FROM THE ORBIT.

By Adolf Alt, M.D.,
ST. LOUIS, MO.

When I received the specimen of bony orbital tumor from Dr. Post it was in a formol solution. It consisted of a somewhat curved thin bony plate with rounded edges 27 mm. long and at its greatest width 13 mm. wide which represented the lamina papyracea of the ethmoid bone. On the nasal side and about at its middle a roundish, partly bony mass was attached which was of the size of a small hazelnut. Its appearance was somewhat brownish. On section it was even macroscopically seen to consist of a number of cells varying in size and partly filled with a chocolate brown coagulated mass. No solid part was visible.

Under the microscope the bony walls of the numerous cells showed nothing abnormal. Nowhere, in the numerous sections, could any signs of corrosion or of proliferation be found. In some sections the small quantity of marrow contained in the thicker parts of the bone was highly infiltrated and in those at the ethmoidal side numerous nests of psammoma bodies were found.

The mucous membrane lining the cells I found in a high state of inflammation. In places it is enormously thickened by hyperæmia and cell infiltration and forms microscopical polypi springing forth into the lumen of the cell; in some parts it is detached from the underlying bone, but this may be an artefact. A large number of mucous glands show a considerable distension of their lumen. The epthelium is well preserved in all the sections.

The specimen, therefore, does not show the presence of any malignant tumor and, more strangely yet, no sign of proliferation or bony growth. The psammoma bodies might, perhaps, point to the presence of an endotheliomatous growth in a part which was not included in the specimen. However, there is no sign of such a growth in all the sections I have examined. The only visible pathological condition is the severe inflammation of the mucous membrane lining the cells of the specimen. The contents of these cells where they have not fallen out are coagulated mucus or blood and mucus.

The only similar condition which I have found described in literature, differs in essential points from our tumor. Hajek (Pathologie and Therapie der entzündlichen Erkrankungen der Nebenhöhlen der Nase, page 214) speaks of hydrops of the ethmoid labyrinth and says: "A process which is very similar to hydrops of the frontal sinus is the accumulation of mucus in the ethmoid labyrinth and the dilatation of its walls. The lamina papyracea is in such cases pressed forward at the inner orbital angle, sometimes rarefied. . . . Thus an oblong swelling is formed which usually fluctuates, unless the bony wall is fully preserved. The eyeball is pressed outward and forward.

The contents were in some cases mucoid and viscous, in others were more fluid and like milk, sometimes of a chocolate color. After an opening was made a cavity was found which had been formed by the confluence of the separate ethmoid cells due to rarefaction of the cell walls.

Whether in these cases there was a retention of mucous, is not certain. It is a fact, however, that in some cases the cavity was lined with a normal mucous membrane, in others by a vascularized membrane, so that there are evidently two different forms of development.

It is unfortunate that in most cases the examination of the nose was not made. We certainly are not wrong in presuming that a dilatation had, also, taken place on the nasal side, as disturbances in breathing are now and then mentioned.

While it is as yet not known definitely whether a dilatation of the ethmoid labyrinth towards the orbit alone does happen, the opposite, such a dilatation toward the nasal cavity alone has been observed."

In Dr. Post's case the nasal examination had been made by a prefectly competent man and nothing pathological had been found in the nose.

The conditions found in the tumor under consideration do not fit Hajek's picture exactly. The tumor was situated considerably farther back in the orbit than the inner canthus. There was no large cavity formed by the confluence of the separate cells by rarefaction of their dividing bony walls. The only pathologic condition the microscopic examination revealed was a high degree of inflammation of the mucous membrane with, perhaps, a retention of mucous, and hæmorrhages into the cell cavities.

A NEW LID CLAMP.

By Anna Ehrhardt.

Nurse in charge Eye Operating Room, New York Eye and Ear Infirmary.

After having seen a number of operations in which excision of the tarsus was performed, it occurred to me that a clamp might be devised which would be of material assistance in making these operations easier of performance. The clamp shown in the accompanying illustration has been in use now for about two years, and it seems to me that it has been of enough aid to surgeons to warrant its publication.

The instrument may be used for either the upper or lower eyelid. In performing resection of the tarsus of the upper lid (Kuhnt's operation) the serrated edge of the instrument is ap-

plied to the conjunctival surface of the lid just above the lid margin and by means of the set screw the lid is made secure. The eyelid is everted and the weight of the instrument is sufficient to hold it in a condition of eversion.

In performing the combined resection of the tarsus and palpebral conjunctiva (Heisrath's operation), or in any other operation, such as excision of the palpebral portion of the lacrimal gland, which calls for double eversion of the upper lid, the eyelid is first put into a condition of single eversion and the serrated edge of the instrument is screwed firmly in place, grasping the conjunctival and sub-conjunctival tissues at the upper border of the tarsus. Then the handle of the instrument is turned upward, and the upper cul de sac is exposed.

TRANSLATIONS.

ON PHOTOPHOBIA.*

By Professor Ernst Fuchs.

Translated by A. Alt, M.D.

Photophobia shows itself as a disagreeable sensation of dazzling, which may be intensified so as to cause real pain, with lacrimation and spastic closure of the lids (blepharospasmus); in many individuals it causes, besides, sneezing. A large quantity of light is not at all necessary to produce the symptoms of photophobia. When awakened from deep sleep at night we open our eyes we can often barely bear the glare of a candle flame. To open inflamed eyes, especially with scrophulous ophthalmia, in semi-darkness is often sufficient to cause blepharospasmus and sneezing.

Blepharospasmus, lacrimation and sneezing are reflex symptoms in which the centrifugal part of the reflex arc is formed by the facial nerve for the blepharospasmus, probably also by the facial for lacrimation, and by the motor nerves of respiration for sneezing. The centripetal part of the reflex arc is either the fifth nerve or the optic nerve.

Irritation of the terminal branches of the fifth nerve is the most frequent cause of photophobia. Among the inflammations of the conjunctiva the scrophulous one (conjunctivitis eczematosa) is especially prone in young individuals to produce so severe a blepharospasmus that it sometimes becomes necessary to narcotize the patient in order to be able to examine his eyes carefully. Yet, just in such cases of particularly severe blepharospasmus the examination often reveals but unimportant and superficial alterations, while in the presence of deep corneal ulcerations the photophobia is not rarely but very slight. In the first case the terminals of the fifth nerve are irritated by the inflammation, while in the second case these have become destroyed. For the same reason, superficial scratches of the cornea, epithelial erosions, as they are called, are more painful and cause more photophobia than deep penetrating injuries. On the other hand even deeply seated inflammations of the cornea, as for instance par-

*Wiener Klin. Wochenschr., January, 1912.

enchymatous keratitis, as also iritis and cyclitis, may cause severe photophobia.

In children with scrophulous conjunctivitis the severe blepharospasmus is a decided obstacle to the healing; the blepharospasm in reality does not infrequently survive the inflammation of the conjunctiva and cornea, having developed, so to speak, into an independent malady. For these reasons it is well not to be satisfied with the cure of the primary affection, but also to combat the blepharospasmus by special means. Since the blepharospasmus is due to the irritation of the terminal fibres of the fifth nerve, the simplest thing is to paralyze these with cocain, and this remedy is consequently applied in order to be better able to examine the photophobic eyes of children. It, also, acts therapeutically, for when the children's eyes are daily strongly cocainized (several instillations of a 5 per cent. solution), they can keep the eyes open a little longer every succeeding day and thus get rid more quickly of the photophobia. The parents, however, must not be ordered to instil the cocain solution several times during the day, for continued instillation of cocain damages the cornea, since cocain kills the cells of the corneal epithelium. Very weak solutions would probably remain useless, especially since through the profuse lacrimation most of it is at once washed away.

An old remedy against the photophobia of children is pouring cold water over their head. Less known is the favorable action obtained in many cases by dionin or suction stasis. Dionin at first causes a sensation of burning in the conjunctival sac which, however, soon disappears; this is followed by hyperæmia and œdema of the conjunctiva, even chemosis, which are gone in the course of a few hours. When a very intense action is desired a little of the dionin powder is put into the conjunctival sac. The suction is a form of stasis introduced by Bier. A little glass cup is put upon the surface of the lids and the air within it is rarefied by a balloon arrangement. This procedure is not painful. After its application the lids and conjunctiva become strongly œdematous. From dionin as well as from this suction I have seen sometimes a rapid diminution of the photophobia especially in cases of parenchymatous keratitis. In both methods of that treatment the œdematous imbibition of the tissues probably acts as an anæsthetic on the fine nerve fibres, perhaps, in the manner of the infiltration of the tissues by Schleich's method in which, too, the œdema is said to play an active role aside from the anæsthetic employed.

Photophobia may, further, be produced by irritation of the terminal fibres of the optic nerve, that is the retina, by light. Here the acting factors are the quantity of light, the diffusion of the light over the retina and the sensibility of the retina.

As to the strength of the light, it is not so much the absolute quantity, which is of importance, as the relative one; this means, when the light is suddenly increased in strength, the proportion to the light in which the eye was previously. When in summer we step out of a dark hall into a place strongly illuminated by the sun we feel a decided photophobia which may increase to distinct pain in the eyes. The blinding influence of a candle light on a person just awaking from sleep, mentioned before, is still more to the point, for the strength of the light of a candle flame is surely very inconsiderable. The sensitiveness of our eye in this direction may be compared to that of the pupil. With a certain illumination the pupil is medium wide. When the light gets suddenly brighter it contracts strongly, only to return gradually to the medium width which it had formerly with the weaker illumination, although the light remains the same. In the same manner the sensitiveness of our eye to light accommodates itself soon to a stronger light so that in a short time this is no longer felt disagreeably.

It is notable that the sudden influence of strong light may produce even pain although neither the retina nor the optic nerve can feel pain. In my opinion the pain is produced by the strong pulling to which the nerves of the iris are subjected when under the influence of bright light the pupil is forcibly contracted. In the same manner the contraction of the pupil under the influence of eserin is accompanied with pain. I conclude that the contraction of the pupil causes these pains from an experiment I made on myself when studying snowblindness and erythropsia. When I stepped from a dark hut out on the sunlit snow I felt a dazzling and pain; the pain, however, did not appear when I had previously dilated my pupils with homatropin, since although the quantity of light entering the eyes was even greater, the pupils could not contract.

After long exposure of the retina to bright light, especially to the light reflected from snow in higher altitudes, erythropsia appears, that is, when one goes from outdoors into a space less illuminated all bright objects, like the windows, a white tablecloth, etc., look a purple red. This phænomenon generally vanishes after a short while. With an exposure to still stronger

light, for instance when looking at the sun with the unprotected eye (as is frequently done when observing an eclipse), the anatomical structure of the retina at the macula lutea is damaged. This may lead to lasting central scotoma with proportional diminution of vision.

It is clear that the eyes must be protected against too bright light. In old times this was done by means of green or blue protective glasses, later by smoke colored ones and in recent times yellow ones have become the mode. In ordering them we are probably not always cognizant of the effect and object of these different protective glasses. The smoke colored ones reduce all visible light rays to an almost uniform degree and restrain, like even colorless glass, a part of the ultraviolet rays. The yellow glasses weaken to an appreciable degree only the shortwave blue and violet ones of the visible rays, but restrain the ultraviolet ones almost completely. Under ordinary circumstances, however, the ultraviolet rays may remain out of consideration. At low altitudes and in ordinary daylight they are very small in quantity, since they are to a high degree absorbed by the lower layers of the air. They are, moreover, not perceived by the retina and can therefore not cause any disagreeable sensations. The disagreeable sensation of dazzling by bright light is produced solely by the visible rays of the spectrum and it is these rays which are damaging to the eye in case of affections of the retina and choroid. In all of these cases, therefore, the ordinary smoke colored glasses are indicated. Only in higher altitudes are ultraviolet rays in large quantities present in the daylight, especially in the light reflected from snow in the mountains, further in the electric light, especially the electric arc light. But in these cases, too, the ultraviolet rays do not as a rule, damage the retina, since they are in a great part restrained by the refractive media of the eye, so that they cannot reach the retina. Their noxious effect is exerted only on the external parts of the eye, the skin of the lids and the conjunctiva which are irritated by the ultraviolet rays and may become inflamed. The inflammation of these parts caused by the light reflected from snow is called snowblindness, the one caused by the electric light electric ophthalmia. To prevent such inflammations yellow (or red) glasses are useful.

The sensation of dazzling may depend, also, on a hyperæsthesia of the retina, so that even small quantities of light are felt disagreeably. This is not rarely the case in neurasthenia and

hysteria and may be compared with the simultaneous hypersensitiveness for noises. In these cases the patients complain not so much of bright daylight as of bright and shining objects. The reflection from the spectacles or their rims renders their wearing impossible, white paper blinds in reading, the white tablecloth while eating. I treated a busy neurasthenic merchant who had finally come to covering the eyes with the hand when he had to sign a paper in order to avoid the most disagreeable dazzling from the white paper.

Dazzling is further complained of when sight is reduced in stronger light. This happens especially in the beginning of the formation of a senile cataract. Such patients bend their head somewhat so that the light does not strike the eye directly and when they want to look more intensely at something they shade the eye with the hand. In these cases the opacity lies in the middle parts of the lens,—usually it forms an irregular opaque disc near the posterior pole of the lens—so that when the pupil is wide light rays can reach the retina, while in brighter light the pupil contracts to such an extent that the opacity covers its area. There is still another quite different condition in which dazzling is complained of, that is in the presence of a central scotoma, as it is seen especially often in chronic nicotin poisoning. Since in all these cases the patients see better at night than in daytime, the condition has been designated as nyctalopia. This can be due to central opacities and is dependent on the width of the pupil or to a central scotoma, when the size of the pupil makes no difference.

The diffusion of the light on the retina, also, is a factor in the dazzling. Under normal conditions the mosaic of the pictures on the retina shows the same sharp outlines of light and dark parts as in the outer world. When, however, diffuse light is spread over this mosaic in a uniform manner vision is rendered indistinct through the diminution of the differences in illumination of the retinal pictures and the sensation of dazzling results. Thus we experience on some days with a uniformly cloudy sky or with a light mist such a dazzling sensation. The cause for such a diffusion of the light lies, however, mostly within our eye. Every opacity in the refractive media of the eye, most frequently, however, those in the cornea, diffuse the light passing through them instead of refracting it regularly. Therefore, it is often possible to improve vision and to remove the dazzling sensation in cases of corneal opacity which occupy only a part

of the pupillary area by rendering this opacity impermeable to light through tatooing it with India ink. The noxious influence of such opacities in the media can easily be demonstrated experimentally. Photograph a printed page first in the ordinary way and then again after having made half of the objective dim with some fatty substance. While the print in the first plate is sharp and distinct, in the second one it is pale and unclean. Now, making a third plate while the fat besmeared part of the lens is covered with black paper, again a clear and sharp picture results.

In very glary light even healthy eyes feel a dazzling sensation because the media in our eye are not absolutely transparent, but always diffuse some light. When the absolute quantity of light is increased, there must, also, result a greater quantity of diffused light. Moreover, in very glary light, some of it enters the eye by the side of the pupil. This is the more so, the less pigment the uvea contains, and especially in albinotic eyes which owe their great photophobia to the fact that much light passes into the eye through the iris and the anterior part of the sclera. The sensation of dazzling in many cases of retinitis may perhaps, too, be due to the diffusion of light, which takes place in the anterior layers of the retina when they are rendered opaque by the inflammatory process.

In the rare cases of total congenital color blindness, too, photophobia is complained of. Once a mother brought me her three color blind daughters stating they behaved like cats, as they became lively and occupied themselves with things at dusk only, while during daytime they were blinded. It is not exactly a photophobia which we find in such cases. Such patients may be put into very bright light without exhibiting the reflex symptoms of photophobia, blepharospasm or stillicidium, but they state that bright light is disagreeable to them, since in bright light they see less. An explanation of this fact has been attempted on the basis of the Perinaud-Kries theory. This theory, as is well known, assumes that the cones in our retina act in perceiving colors and in seeing in bright light, while their function is to a certain extent restrained with diminished light. The rods, however, serve for vision in weak illumination and are less and less sensitive as the light increases. From this our eyes see in bright light in the main through the cones, in weak light principally with the rods. The total color blindness in these cases then would be due to the lack of cones, according to

this theory. Since such patients see by means of rods only, their eyes are especially adapted for seeing at dusk, and in weak light they see like normal eyes, which in that case, too, see by means of the rods only. In bright light on the other hand the rods functionate little or not at all, thus the totally color blind individual sees less and has the sensation of being blinded. According to some the color blind is said to be absolutely blind in very bright light.

NOTICE.

PAPYRUS EBERS.

Some time ago, announcement was made through the pages of this journal that Dr. Carl H. von Klein had completed an English translation of the Papyrus Ebers, and that its publication in book form depended upon an advance subscription list of one thousand names. As a result of that announcement and the interest shown by Dr. von Klein's friends, six hundred subscriptions have been secured. The enterprise drags at this point and something must be done to arouse the profession to the importance of preserving this valuable manuscript. Four hundred additional subscriptions must be secured.

We sincerely hope that the ophthalmic public to whom the Papyrus Ebers is of especial interest will respond to this appeal for further subscriptions, which should be sent to Dr. C. H. von Klein, Medical Department, John Crerar Library, Chicago, Ill.

MEDICAL SOCIETIES.

THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Thursday, November 9th, 1911.

Mr. J. B. Lawford, President, in the Chair.

ELECTION OF HONORARY MEMBERS.

The President announced that some time ago the Council determined to propose the names of three gentlemen for Honorary Membership: the late Dr. Huhglings Jackson, Sir Jonathan Hutchinson, and Dr. Landolt, the Bowman Lecturer of last summer. The result of the ballot was the unanimous election of Sir Jonathan Hutchinson and Dr. Landolt.

CARD SPECIMENS.

Mr. Foster: A case of bilateral angioma of retina.

The President asked whether Mr. Moore thought the title was quite accurate. He thought that what could be seen were unusually dilated retinal vessels, but not newly-formed vessels. Mr. Foster Moore replied that there was a very large artery going to it, and a large vein coming from it.

Mr. Hugh Thompson: A case of second orbital endothelioma occurring 11 years after removal of the first. He said he regarded it rather as a fresh growth than as a recurrence of the former one.

Mr. J. Cole Mitchell: Three cases of rodent ulcer which had been successfully treated by means of carbon dioxide snow. The President regarded them as very good examples of the successful use of the method.

Mr. N. Bishop Harman: A case with dislocation of the lens in the posterior chamber due to an accident 18 months before. The vision had remained excellent.

Mr. Wray showed a new model of a Placido's disc, and retinoscopic lenses.

PAPERS.

The Pathology of Dust-like Opacities in the Vitreous Body and of Descemet dots.—Professor Straub, Amsterdam.

Professor Straub said that among the diseases of the eye there should be recognized an inflammation of the vitreous body which he proposed to name "hyalitis." He injected into the vitreous body of the rabbit some pathogenic microbes. These microbes grew there and there only, and attracted to the vitreous body serum and leucocytes. The serum and leucocytes were produced by the vessels of the ciliary body, but that was not a reason to talk of cyclitis in cases of inflammation of the vitreous body. He likewise injected into the ciliary body pathogenic microbes, and to those went serum and leucocytes, and there was produced a real cyclitis, quite different from the hyalitis which he had in the first experiment. In the laboratory hyalitis could be distinguished from cyclitis, and the same distinction should be made in practical work. But that had not yet been done. The two diseases were mixed up in the text books. The books said the ciliary body poured out its exudate into the vitreous body, but that was a mistake, as leucocytes did not allow themselves to be poured out. Metchnikoff said that leucocytes only went where they were attracted by chemotactic substances. When there were microbes in the vitreous body, those substances went towards the vitreous body; when microbes were in the ciliary body the leucocytes went to the ciliary body. He had, in order to test the matter, tried to produce inflammation of the ciliary body, by infecting the ciliary body with tubercle bacilli, causing in that way granuloma. He then made sections to see whether the vitreous was clear. He thought it was clear, with the exception of a few leucocytes here and there. But when making sections through the eye he found that the optic nerve was somewhat swollen, and that in its cup there were many leucocytes, and those were projected on to the immediate adjacent retina. So he concluded that tubercular granuloma in the ciliary body had the power to cause a very slight inflammation of the optic nerve on the other side of the eye. The lymph stream in the eye went from the ciliary body to the optic nerve, and took with it some toxins which were produced by the granuloma. Those experiments were made 20 years ago. He did not feel satisfied with his method of infecting the ciliary body, therefore 8 years ago he recommended, with a colleague, a new series of experiments, in

which a thread charged with tubercle was passed through the ciliary body, through the sclerotic, and through the superficial layer of the ciliary body, and brought out into the sclerotic again. The thread was cut at the points of entrance and exit, so that there remained in the ciliary body a piece of thread 3 mm. long, and after 10 to 14 days a granuloma commenced. Some weeks later the eye was excised and sections were made, and he found the same things as in his experiments 20 years ago. He showed colored photographs, exhibiting the points he had mentioned. The dots were caused by groups or heaps of leucocytes. Among 14 cases of dust-like opacities only one was syphilitic, 9 were tubercular, and the remainder he did not know the nature of. It was quite clear that Descemet dots were groups of leucocytes. The vitreous body, he felt sure, obtained no leucocytes from the cornea; in inflammation of the vitreous body the leucocytes came from the vitreous body.

The President thanked the Professor for his able exposition, based upon prolonged and numerous experiments. Mr. Treacher Collins asked how the leucocytes which died got to the back of the cornea in the first place. Mr. Herbert Parsons said he understood Professor Straub's contention to be that the leucocytes were carried forward in the lymph stream, and his explanation of the formation of the dots was a very ingenious one. But the speaker criticized the use of the term "hyalitis" and "descemetitis," as he regarded their use as backward steps in pathology. The tissue under discussion was non-vascular and apparently passive, whereas inflammation was pre-eminently an active process. He had hoped the Professor would have supported the view which he, Mr. Parsons, had hypothecated, that where there were dust-like opacities in the vitreous associated perhaps with choroidal change there was really a low grade of cyclitis going on to account for the presence of those opacities, and that they were not due to any inflammation present in the vitreous *per se*. Supporting that view was the fact that in cases of choroidal inflammation there was no transference of leucocytes or organisms directly from the choroid into the vitreous until the membrane was burst through. Mr. W. H. Jessop said the Professor did not appear to have made quite clear what he understood to be the difference between hyalitis and cyclitis. He had been much interested in the demonstration of leucocytes in the cup of the optic nerve, and the slight degree of papillitis. That was particularly interesting because one often felt there must be some papillitis because of the degree of swelling.

Professor Straub replied, affirming his belief in his ultimate power to convince ophthalmic surgeons of the truth of his thesis when he came to publish the full results of his work. He relied for the acceptance of his views upon a combination of the histological and clinical material which he had collected.

Ocular Conditions found in Mongolian Idiots.—Mr. A. W. Ormond.

He had examined a number of these cases at Earlswood and Darenth Asylums, at the Evelina Hospital, and elsewhere, in all 43 cases. He described the facial and physical traits. They had a certain liability to particular diseases. Over 50 per cent. had a defect in their lenses, and almost all had some ocular defect. Sometimes Mongolians could be recognized as early as the second year. When seen later they were short of stature, due mainly to the shortness of the limbs. The head was round and the occipital protuberance of the skull ill-developed. The hands were small and the thumbs squat, the little finger being incurved. The foot was flat, and the subjects often sat tailor-fashion. They had a difficulty in pronouncing certain consonants. They were imitative, fond of music and affectionate. Most of them died from tuberculosis, and they did not often attain adult life. There was almost constantly some ocular trouble present—blepharitis, ectropion, squint, nystagmus, or lens opacity. Blepharitis and conjunctivitis might be primarily due to dirty habits, and might be kept up by uncorrected errors of refraction. A more certain cause of the inflammatory condition of the lids was a dry, glazed condition of the skin of the lower lids, which, by its contraction, caused a slight degree of ectropion. In more than 50 per cent. of his cases, some form of lens opacity was present, and with such a high proportion, it might be regarded as an aid to diagnosis. The cataracts were of the incomplete form, and most of them of the "dot" variety, in the position common for lamellar cataract. These dots, when slight were often translucent, and so could not be seen by transmitted light. The opacities did not reach to the periphery of the lens in any direction, and consisted of numerous small discrete dots. The posterior pole of the cataract was often marked by a star-shaped opacity. Though the teeth of these people were defective, they did not show the honey-combed condition so frequent in cases of lamellar cataract. Mr. Ormond could not record accurately the visual acuity, as the children were not sufficiently controllable to be

trusted with glasses. The youngest of the patients showing cataract was aged 6½ years, and the oldest 43. Mongolian imbeciles were, in many cases, the children of old parents, or the last child in a large family. Of his 42 cases, 32 were males, and 10 females. The average was 14¾ years. Twenty-three had the interpalpebral fissure directed upwards and outwards, 5 had nystagmus, 9 had squint, 18 had either blepharitis or ectropion, or both, 11 had epicanthus, and 25 had some lens opacity.

Thursday, December 14th, 1911.

Mr. J. B. Lawford, President, in the Chair.

Dr. Edridge Green showed a new simple test for color blindness, and Mr. C. D. Marshall regarded it as a great advance in accuracy of testing, as it provided for all the colors which a candidate could be expected to name. Mr. A. H. Levy showed a case of exudation into the canal of Cloquet, and one of dystrophia adiposo-genitalis with optic atrophy, and the latter was discussed by Mr. J. H. Fisher and the President. Mr. W. H. McMullen exhibited a case of nearly complete congenital external ophthalmoplegia. It was discussed by the President, Mr. Parsons, and Mr. Bishop Harman. Mr. A. S. Worton showed a punctate crystalloid deposit in both corneæ and Mr. Bishop Harman a case diagnosed as retinitis circinata, but in which the retinal exudation disappeared in a year. The President, Mr. Doyne, Mr. J. H. Fisher, Dr. Edgar Stevenson (Liverpool) discussed the condition. Mr. Grimsdale showed a case of cyst of iris. Mr. Greeves showed a case of sixth nerve paralysis in a child after acute polioencephalitis. The President and Dr. Gordon Holmes discussed the case. Mr. McMullen showed an example of retinal disease with massive exudation. Mr. Doyne and Dr. Harrison Butler discussed the case. Mr. Lister showed a pigmented conjunctival growth. Mr. Hewkley brought forward a case of punctured wound of the cornea, with edges of wound stained with ink. Mr. C. D. Marshall showed, for Mr. Ridley, a model of a scotometer.

Dr. Thomson Henderson read a paper entitled "The Pathogenesis of Choked Disc." Dr. Thomson Henderson said that the brilliant results obtained by cerebral decompression had con-

clusively proved that the manifestations of choked disc were the direct expression of the mechanical agency of increased intra-cranial pressure. In choked disc the intra-ocular pressure was normal while the intra-cranial pressure was raised, and therefore to appreciate the essential relationship between cause and effect it was essential for us to possess a clear and definite conception of the physiological association between the normal intra-ocular pressure and the normal intra-cranial pressure. He had experimentally demonstrated that under physiological conditions the intra-ocular and intra-cranial pressure were similar in nature and in level. The condition of venous engorgement and of swelling of the disc, which together completed the opthalmoscopic picture of choked disc, were both the result of the sudden break at the level of the lamina cribrosa of the physiological equilibrium of pressure which normally existed at this point. When the intra-cranial pressure was raised, the cerebral venous pressure mounted to the same level, and therefore the pressure in the neural portion of the retinal vein likewise rose, hence, to complete the retinal circulation, the pressure in the intra-ocular portion had to rise *pari passu,* thereby producing the retinal venous engorgement. As fluids tended to lie at the lowest hydrostatic level, and as the hydrostatic pressure behind the lamina cribrosa was greater than in front, fluids passed forward into the now lower hydrostatic level of the eye, and so caused swelling of the disc. The arching forward of the lamina cribrosa, and the so-called hydrops vaginæ represented a yielding of those structures to the increased hydrostatic pressure they were called upon to support. As the brain acted as a viscous and not a fluid mass, and further, as the rigidity of the falx and tentorium tended to hinder general diffusion of hydrostatic pressure, the pressure in one cerebral hemisphere and corresponding optic nerve might rise above that in the other, thereby accounting for the ipsolateral feature of choked disc. The paper was discussed by Mr. Herbert Parsons, who considered that on logical grounds Mr. Henderson's contention did not hold good. He believed that pressure was not a question of volume in the sense in which Dr. Henderson meant it. If the intra-ocular pressure were identical, physically, with intra-cranial pressure, then admittedly the intra-ocular pressure would be equal to to the intra-ocular venous pressure. For the intra-ocular pressure to be always equal to intra-ocular venous pressure would mean absolute rigidity of the cornea and sclera. That was where the fallacy

lay. There was evidence that the sclera was not quite rigid. He did not believe a purely physical explanation could be given either of glaucoma or of increased intra-cranial pressure; physiological factors must be taken into account. Mr. J. L. Paton regarded Dr. Henderson's paper as a return to the crude original ideas published in the sixties, only modified by an application to the explanation of venous congestion published by Dr. Leonard Hill in 1896. Venous pressure alone did not suffice to account for the phenomenon of choked disc, and in this connection he mentioned the example of thrombosis of the central vein. He asked the author in what proportion of the cases of optic neuritis, which he had examined, he had seen the arching forward of the lamina cribrosa. Out of 60 eyes in which there was optic neuritis in only one was there that arching forward of the posterior fibres of the lamina cribrosa. Dr. Gordon Holmes thought it remarkable that when views such as these in this paper were put before a scientific society, the facts also were not given. He proceeded to discuss the question of the disassociation between intra-optical and intra-cranial pressure, and regretted that the question of ipso-laterality had not been considered more quietly.

Dr. Adolph Bronner read a paper entitled "Notes on Three Cases of Ulcer of the Cornea Combined with Painful Spasm of the Sphincter Pupillæ." In the first case the severe pain persisted, in spite of all ordinary methods of treatment, from October 9th to November 21st, when an iridectomy was performed, which gave immediate relief, and the ulcer healed in four days. In the second case the symptoms persisted from June until August, when an iridectomy again gave immediate relief. The third case persisted for three months and was at once cured by iridectomy. The peculiarity of these cases was that the ulcers were superficial, often traumatic, not infiltrated, and did not heal in spite of the usual methods of treatment, for several weeks or months. The pain and photophobia were also more severe and and more intermittent than usual, and at once ceased after iridectomy. The chief peculiarity, however, was that that pupil did not dilate with atropine, and that often the pupil of the other eye did not act as readily as usual. The fact that the iris was not infiltrated, that there was no lymph on the anterior capsule, and that the pupil dilated after iridectomy, proved that there had been no severe iritis. Probably the unusual symptoms were due to neuritis of the nerve endings in the cornea, which caused reflex spasm of the constrictor pupillæ muscle, and this, in turn,

possibly prevented the ulcer from healing. The President said the treatment seemed rather like a return to the methods of former days. He recounted the case of a gentleman who had been under the care of several ophthalmic surgeons for corneal ulcer, but without success until the patient went to the late Mr. Gunn, who did an iridectomy, and the patient promptly got well. Mr. Richardson Cross suggested the use of the cautery and tapping the eye, before making the radical operation. Dr. Harrison Butler said that in almost every serious case of ulcer which he did not cure by other means he obtained a favorable result by the cautery. Dr. Bronner replied that he used the cautery in one case but it did no good. Of course he did not suggest that iridectomy should be done in every case of corneal ulcer. The cases he had related were not septic ones.

ABSTRACTS FROM MEDICAL LITERATURE.

By J. F. Shoemaker, M.D.,

St. Louis, Mo.

SUBHYALOID AND VITREOUS HÆMORRHAGES.

A STUDY OF POSSIBLE SYSTEMIC CAUSES.

Hiram Woods (*Jour. A. M. A.*, July 29, 1911), in the study of this subject, made inquiries of a large number of ophthalmologists as to their experience with this condition and their views as to its cause; from the replies received and his own experience he makes the following deductions:

1. Results.—So far as the above observations admit of analysis, from the standpoint of results, the following is approximately true: Of forty-eight eyes, twenty recovered with practically normal vision to the time of last observation. Seven obtained more or less improvement, the nature of the residual lesion not being given. Seven developed retinitis proliferans, three of these being totally blind, the others preserving sight enough to do some work. The other fourteen were totally lost, four from secondary glaucoma, one from phthisis bulbi, and nine from causes not specified, probably vitreous opacities, retinal deachment, etc. Seventeen out of the forty-eight eyes were lost.

2. Recoveries.—A number of cases in the foregoing reports and in literature, showed one hæmorrhage, or recurrent hæmorrhages, producing temporary and complete blindness. Then normal vision was slowly restored and retained. Nothing was found in the eye to account for hæmorrhage. The source of blood is generally ascribed to the choroidal vessels. This is based on the facts that the retinal vessels, when they can be seen, as in the subhyaloid variety, show nothing wrong, and after absorption of the blood, the same is true. It is hard to think of normal retinal vessels being responsible for so much hæmorrhage. A still stronger reason is found in our knowledge derived from recent studies in choroidal diseases. We know that many pathologic conditions are associated with exudative choroiditis. Intestinal parasites, "indigestion," chronic appendicitis, and malaria, may be mentioned. We attribute these ocular lesions to toxins or organisms in the blood, and their manifestation in the choroid to the large vascular area therein. It is not going very far to imagine the same influences breaking down a vessel and producing hæmorrhage. So long as vascular resistance is less than the acting cause, recurrence persists. When the cause is removed, through either intelligent diagnosis and treatment or general hygienic management, hæmorrhages cease. Note, for instance, the cases reported by Greenwood and Bordley. Recovery of vision must depend on the harm done by one or more attacks, and recurrence must depend on intelligent or accidental removal of the cause.

3. Concomitant Systemic Conditions.—Blood-pressure. Black's remark is apparently borne out by the "returns." When high, it is usually associated with nephritis or arteriosclerosis. In each we have conditions directly calculated to produce hæmorrhage through a previous arteritis or phlebitis. Bull believed that such patients always develop arteriosclerosis or nephritis, if they live long enough. One of my own patients was dead in a year of nephritis; yet no one would have made such a diagnosis on this young woman on the urinalysis and symptoms alone—outside of the intra-ocular hæmorrhages and blood-pressure. C. S. G. Nagel reports a case in which retinal hæmorrhages were the first symptoms of diabetes. (I call attention here to the previously expressed opinion that we must not differentiate intraocular hæmorrhages too closely according to their situation.) The patient was apparently in good health. Nagel notes Leber's observation that retinal diabetic lesions occur only after years, and

when emaciation and general decline are present. Webster Fox discusses the relation of high pressure to intra-ocular hæmorrhages, and shows the close relation between them and various forms of nephritis. In the article by Scrini and Bourdeaux, the statement is made that these essential vitreous hæmorrhages have no prognostic effect on general health. I think that this statement is to be accepted with allowance. Nagel's diabetic, and my first case are illustrations. My third belongs to the class which must be regarded with concern. Something caused the high arterial tension, and the fact that we do not know what it was in no way proves that it is not serious. I cannot extend the paper to quote other authors, but I am convinced that these hæmorrhages, accompanied by high pressure, ought to be regarded as a possible early indication of chronic nephritis. But they occur, too, with low pressure. Black expresses the opinion that dilated heart or auto-infection is at the bottom of these. We still have the dilated heart to account for. Usually some systemic infection precedes this. Of course, the passive venous congestion might be a mechanical cause. As to auto-infection, i.e., absorption of either the organisms themselves or toxins, which might act deleteriously on small or terminal vessels, I believe that here we are approaching the main ætiologic factor in most cases, and that careful search for the nature of the infection may often lead to the diagnosis of a condition giving no other symptom.

Axenfeld and Stock report a case of a woman, 30 years of age, with a vitreous hæmorrhage of the right eye. The left eye was normal; but several weeks later a grayish-yellow exudate appeared in the periphery of this eye. There was also a perivascular exudate along a retinal artery which ran toward the choroidal focus. There was both general and local reaction to tuberculin in both eyes. "During the treatment with small doses of tuberculin the foci in the choroid and perivascular exudate completely disappeared, and in the right eye, in which in the meantime an indolent posterior synechia appeared, the vitreous cleared. One must accept that in this case a chronic tuberculous uveitis had begun with the appearance of a simple hæmorrhage in the vitreous, which surely had arisen from the retinal vessels." It seldom happens that one can observe such a sequel of events in these cases.

De Schweinitz and Holloway report a remarkable case of retinal hæmorrhages in a boy of 20, who gave a positive reaction

for tuberculosis. The late Dr. Kipp, of Newark, reported an instructive case in this connection. I call attention to my fourth case, which showed positive tuberculous reaction without symptoms outside the eye. Such observation seems to me to show that these cases, even if the eye gets well, must always be viewed with suspicion, and that it is best to treat them as though the diagnosis was made. Greenwood's policeman (given in this paper), and Bordley's sixth case are examples.

Indicanuria plays a large rôle in these concomitant systemic conditions. There is, I know, discussion regarding its value as a diagnostic sign; but its persistent presence in excess is generally thought indicative of disturbance of metabolism. It was observed in most of Bordley's and in Bulson's cases. It was seen in my series, but not in sufficient quantity to make us think it significant. In another it was abundant.

The frequency of obstinate constipation in the description of these cases leads one to think that indican would be found more frequently if always looked for. It occurs without reference to blood-pressure. Where the pressure was high, as in Bordley's cases, there were other conditions, usually renal, to account for it. The close association of indicanuria with functional and organic uveal lesions has been presented to this Section too recently to need repetition. Vitreous hæmorrhage, with tuberculosis (sometimes clinically undemonstrable) shows that this form of infection can produce vascular diseases in the eye. Indican in the urine means intestinal putrefaction, and this means not necessarily absorption of putrefactive toxins, but malassimilation, allowing absorption of other toxins, which, under better conditions, the system might avoid. Here we come against other concomitant conditions. Anemia, unless essential, must have a cause. Dysmenorrhea, amenorrhea, unless due to local conditions, often admit of an explanation which involves nutrition. They are but symptomatic of the same disturbance which causes the intra-ocular hæmorrhage, and are not themselves a cause.

To discuss treatment of these hæmorrhages is impossible, save to say that for the most part oculists have been using diaphoresis, the iodids, etc. With the natural tendency to absorb, it is hard to estimate the value of these remedies. It seems to me that we should view these cases about like this:

1. In older people, i.e., persons over 50, we usually have signs of arteriosclerosis, and if they are not present, the hæmorrhage itself is evidence. Eliminating special causes of vascular changes—syphilis, nephritis, etc.—we have to manage, as best

we can—or the internist has to manage—the vascular changes. Along this line, we shall get our best results.

2. In young people, whether there is a single hæmorrhage or many, we are probably dealing with some infection or disease capable of producing vascular changes. High blood-pressure throws suspicion on the kidneys, whether or not there are other symptoms of nephritis. Anemia, menstrual disturbances, etc., are but symptomatic of malnutrition probably due to an infection. This may be tuberculous, and may be of some other kind, brought about through defective metabolism. Its presence and nature is to be determined by rigid laboratory examinations.

3. Even when we can find no cause, and have to resort to the "alterative" treatment, we should bear in mind that the cause has not been determined; that it is usually of a serious nature, and that it is well to give the patient the benefit of doubt with regard to the three causes apparently most apt to be found—tuberculosis, defective metabolism, nephritis.

BOOK REVIEW.

A Pocket Atlas and Text Book of the Fundus Oculi, with note and drawing book. By G. Lindsay Johnson, M.A., M.D., F.R.C.S., and Arthur W. Head, F.Z.S. Chicago. F. A. Hardy & Co. Price $2.50.

This is an American edition of the pocket atlas which we have had occasion to recommend to our readers in a former issue, when it was published in England. Dr. Casey A. Wood wrote a preface to this American edition. We gladly again recommend this handy book, especially to students to whom it will prove a safe and useful guide into the realms of ophthalmoscopy. Alt.

NOTICE.

It is with the greatest regret that we hear that the Editor of the Ophthalmic Year Book has decided to abandon its publication for this year and, unless more interest is shown by the ophthalmological colleagues, perhaps altogether.

More interest means more subscribers. Everyone acquainted with the excellent work the editor and his co-editors have given us in these Year Books should try and help in making its continuance possible and worth while.